Late blight (*Phytophthora infestans* (Mont) De Bary) development from potato seed-pieces treated with fungicides

William W Kirk,* Brendan A Niemira, Jeffrey M Stein and Raymond Hammerschmidt

Department of Botany and Plant Pathology, Michigan State University, East Lansing, MI 48824, USA

Abstract: Fungicides were applied as seed-piece treatments to control potato late blight, caused by *Phytophthora infestans*, US8, A2 biotype in controlled environment and field experiments. Efficacy of seed treatments for controlling late blight was examined under three disease development regimes simulated by artifical inoculation; (a) seed-borne infection, (b) transmission of infection resulting from spread during the seed-cutting operation, and (c) infection of foliage by aerial inoculation. Emergence of plants from the seed-borne infection was uniformly low (<40%) in controlled environment and field experiments. In controlled environment experiments some of the plants that emerged from fungicide-treated seed-pieces were infected with late blight. Following exposure of tuber surfaces to *P infestans*, emergence rates from seed-pieces treated with formulated products that included mancozeb in the formulation were comparable to the untreated and non-inoculated control in controlled environment and field experiments. Plants that emerged from non-inoculated seed-pieces treated with fungicides that contained active ingredients known to be effective against foliar late blight had lower percentage foliar infection after inoculation than the untreated control. Leaves close to the base of the stem had fewer infections than leaves attached at the mid region of the main stem, 14 days after inoculation, in some of the controlled environment studies. In contrast, field experiments conducted under conditions conducive to late blight development showed that none of the seed treatments applied to late blight-free seed-pieces delayed the onset and severity of late blight infection. In potato production areas at risk of early season late blight, seed treatments applied to healthy seed may confer limited protection against late blight between planting and the first scheduled applications of prophylactic foliar fungicides.

Keywords: fungicide; *Solanum tuberosum*; tuber; seed-borne disease; emergence; *Phytophthora infestans*

1 INTRODUCTION

Potato late blight caused by *Phytophthora infestans* (Mont) De Bary is the most important potato disease in North America.[1] Control of late blight is traditionally achieved by cultural controls and crop-protection strategies that rely on applications of foliar fungicides.[2] Late blight is readily transmitted by seed-borne inoculum[3,4] and, consequently, immature stems and leaves may be exposed to late blight from infected seed-pieces. Other seed-borne pathogens of potatoes, such as *Fusarium* spp and *Rhizoctonia solani* Kuhn, can be effectively controlled by application of fungicides to the seed-piece.[5,6] Similarly, prevention of establishment of late blight infection transmitted from infected to disease-free seed-pieces by seed treatments has been demonstrated.[7] Active ingredients in seed-piece treatments may prevent early-season establishment of late blight by persisting on the immature foliage after the plant has emerged from the soil.[8]

The objectives of this study were to determine the efficacy of fungicides applied to potato seed-pieces against potato late blight under different infection mechanisms. Experiments in controlled environments and in the field were conducted between 1996 and 1998 to establish the efficacy of a variety of fungicides applied to the seed-pieces in controlling tuber-borne and foliar phases of late blight. Three separate simulations of late blight infection mechanisms and potential prevention of late blight establishment are described: infection carry-over within tubers stored for seed and infected during seed cropping in the prior growing season, transmission of late blight between

* Correspondence to: William W Kirk, Department of Botany and Plant Pathology, Michigan State University, East Lansing, MI 48824, USA
E-mail: kirkw@pilot.msu.edu
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infected and non-infected seed-pieces during seed cutting, and infection of the immature canopy after exposure to aerial inoculum.

2 MATERIALS AND METHODS

2.1 Selection and preparation of potatoes

Seed tubers of potatoes (*Solanum tuberosum* L cv Snowden used in 1996–1997 and cv Onaway used in 1998) harvested from crops in Michigan with no record of foliar or tuber late blight were selected for freedom from other diseases and uniformity of size (35–55 mm diameter size grade). After harvest, the tubers were stored at 4 °C in the dark for at least 80 days, then transferred to 18 °C, (14 h photo-period) to break dormancy.[9] Prior to storage the tubers were washed in distilled water, disinfected for 2 h in 20 g liter^{-1} commercial bleach solution (Clorox), dried and stored until used. The number of leaf initials at the time of seed treatment was established by counting the number of leaf initials on sprouts from apical, lateral and basal regions of the tuber under a dissecting microscope ($n = 20$ sprouts). Seed treatments were applied after inoculation. The timings of cutting of seed tubers into seed-pieces and application of seed-piece fungicides were dependent on the simulated infection mechanism and are described in the following sections. Seed-pieces were planted 48–72 h after the seed treatments were applied.

2.2 Experimental design

Experiments testing infection transmission mechanisms from seed-pieces infected during the seed-production season and infection of the immature canopy were conducted during each of three runs in controlled environments and in the field in 1997. In addition to these two infection mechanisms simulated, experiments testing transmission of infection from infected seed-pieces to late-blight-free seed-pieces were run in 1998. The controlled environment experiments were carried out in temperature- and humidity-controlled environment chambers. The chambers (3.4 m^3) were situated within greenhouses and covered with 1 mm transparent polyethylene. Natural light was supplemented by high-pressure sodium lamps, 400 W 14–10 h day–night. Relative humidity was maintained at greater than 90% by timer-controlled humidifiers (Herrmidifier model 500). Temperature typically ranged between 15 and 24 °C. Ten plants per treatment were used for each experiment (1997 and 1998) in concurrent experiments.

2.2.1 *Seed tuber infection simulation and inoculation*

This study examined the effect of seed treatments on suppressing transmission of *P infestans* from infected seed tubers to emerging sprouts. Tubers were inoculated with a zoospore suspension (10^3 zoospores ml^{-1}) of *P infestans*, US8 genotype (insensitive to metalaxyl, A2 mating type) from cultures grown on rye agar plates[10] for 14 days in the dark at 15 °C. Sporangia were harvested from the Petri dishes by rinsing the mycelial/sporangial mat in cold (4 °C) sterile, distilled water and scraping the mycelial/sporangial mat from the agar surface with a rubber policeman. The mycelial/sporangial suspension was stirred with a magnetic stirrer for 1 h. The suspension was strained through four layers of cheesecloth and the concentration of sporangia was adjusted to about 1×10^6 sporangia liter^{-1} using a hemacytometer. Tubers were inoculated with about 0.1 ml of the zoospore suspension (*c*100 zoospores) injected to a depth of 2.0 mm below the periderm and 1.0 cm from the main apical sprout. The wound was covered with a smear of petroleum jelly to prevent desiccation. Seed treatments were applied 24 h after inoculation.

2.2.2 *Transmission of infection at seed-cutting simulation*

Transmission of late blight infection at seed cutting was simulated by cutting seed and immediately exposing the seed to late blight inoculum. The seed tuber was cut into two pieces with a sterile knife. The exposed cut surface was placed face down on a 14-day-old, homogenized mixture of mycelium and sporangia of *P infestans* in rye agar for 30 s, removed and seed treatments were immediately applied. The homogenate was prepared from 20 plate cultures (9 cm diameter $\times$ 15 mm depth Petri plates). Each plate produced between 10^5 and 10^6 spores ml^{-1} from 50 ml of wash water. An estimate of the amount of mycelium from each plate was not attempted.

2.2.3 *Foliar infection simulation and inoculation*

The effect on foliar infection of seed treatments applied to non-infected tubers was examined. Foliar late blight was evaluated on plants inoculated with late blight after the plants had emerged. After they reached the rapid expansion phase (15 days after emergence, 15 cm tall with an average of 10 main leaves on each stem), the plants in the chambers were inoculated with 1000 ml of a 10^3 zoospore ml^{-1} suspension, delivered as an aerosol. Plants were exposed to high relative humidity conditions (RH = 100%), for 8 h prior to inoculation to wet the leaves. In the field experiments all the rows were inoculated (100 ml (12 m)$^{-1}$ row) with a zoospore suspension (10^3 zoospore ml^{-1}) on 30 July 1997 (experiment 1) and 23 July 1998 (experiment 2).

2.2.4 *Application of seed treatments (controlled environment and field experiments)*

Conventional seed treatment fungicides were applied to whole seed for use in the transmission of late blight from seed harvested the previous season. Seed tubers cut once were used for experiments to examine transmission during seed-cutting and protection of the immature canopy from aerial inoculum. Treatment trade names, manufacturers and chemical compositions are listed in Table 1. Application rates are listed in Tables 2 and 3. Seed treatments were

Table 1. Fungicides applied: trade names, manufacturers and active ingredients

Trade name	*Manufacturer*	*Active ingredient*
Dithane 75DF	Rohm and Haas	Mancozeb, 750 g kg^{-1}
Maxim MZ 0.5D	Novartis	Fludioxinil 5 g kg^{-1} + mancozeb 60 g kg^{-1}
Maxim 0.5D	Novartis	Fludioxinil 5 g kg^{-1}
Tops MZ/Curzate	Gustaffson	Thiophanate-methyl 25 g kg^{-1} + mancozeb 60 g kg^{-1} + cymoxanil 10 g kg^{-1}
Tops 5 5D	Gustaffson	Thiophanate-methyl 50 g kg^{-1}
Tops MZ 8.5D	Gustaffson	Thiophanate-methyl 25 g kg^{-1} + mancozeb 60 g kg^{-1}

Table 2. Controlled environment experiments: effect of seed treatments on emergence rate (RAUEPC), final number of plants emerged (%), percentage of emerged plants infected with *Phytophthora infestans* and average amount of foliar blight over the infection period (C only)

	Inoculation type															
	A *Injection*				B *Cut face and dip*						C *Foliar*					
Treatment, formulation and rate of application (*kg tonne*$^{-1}$)	*RAUEPC*[a]		*Plants emerged (%)*		*RAUEPC*		*Plants emerged (%)*		*Plants with lesions (%)*		*RAUEPC*		*Plants emerged (%)*		*RAUDPC*[b]	
TPM[c] 2.5DS (10)	0	b[d]	0	b	22.5	a	40	a	20	a	47.1	ab	90	a	29.1	b
TPM + mancozeb 8.5DS (5)	0	b	0	b	56.3	a	100	a	0	a	47.6	ab	100	a	29.8	b
TPM + cymoxanil + mancozeb DP (5)	0	b	0	b	35.0	a	80	a	20	a	41.6	ab	89	a	30.7	b
Fludioxinil 0.5DS (5)	2.3	b	18.3	b	33.8	a	60	a	20	a	47.4	ab	99	a	32.6	b
Fludioxinil + mancozeb 1DS (5)	12.5	b	40	b	33.8	a	60	a	0	a	28.0	ab	80	a	30.9	b
Untreated/non-inoculated	53.7	a	100	a	56.3	a	100	a	0	a	53.7	a	100	a	0	a
Untreated	0	b	15	b	22.5	a	40	a	20	a	47.8	ab	90	a	36.2	b

Potato seed-pieces were either (A) injected with a zoospore suspension of *P infestans* then treated with seed-piece fungicides 48 h after inoculation (average of six replicated experiments); (B) cut and inoculated by placing the cut surface onto a mycelial homogenate of rye agar media and *P infestans* average of three replicated experiments); (C) Potato plants were inoculated with an aerosol spray of a zoospore suspension of *P infestans* (average of six replicated experiments), 1996–1998.

[a] RAUEPC, relative area under the emergence progress curve estimates the rate of emergence. Higher numbers equate to a greater rate of emergence. Calculated from day of planting to eight days after planting with maximum = 100.

[b] RAUDPC, relative area under the disease progress curve calculated from the day of inoculation to 21 days after inoculation with maximum = 100.

[c] Thiophanate-methyl.

[d] Values followed by the same letter are not significantly different at $P = 0.05$ (Tukey Multiple Comparison).

Table 3. Field experiments: effect of seed treatments on emergence rate (RAUEPC), final number of plants emerged (%), percentage of emerged plants infected with *Phytophthora infestans* and average amount of foliar blight over the infection period (C only)

	Inoculation type															
	A *Injection*				B *Cut face and dip*				C *Foliar*							
Treatment, formulation and rate of application (*kg tonne*$^{-1}$)	*RAUEPC*[a]		*Plants emerged (%)*		*RAUEPC*		*Plants emerged (%)*		*RAUEPC*		*Plants emerged (%)*		*RAUDPC*[b]	
TPM[c] 2.5DS (10)	8.4	b[d]	22.0	b	31.4	c	61.7	bc	51.4	a	96.5	a	33.7	b
TPM + mancozeb 8.5DS (5)	6.1	b	19.0	b	57.8	a	98.3	a	52.6	a	96.0	a	32.3	b
TPM + cymoxanil + mancozeb DP (5)	7.3	b	23.7	b	54.8	ab	93.3	ab	52.3	a	97.0	a	32.7	b
Fludioxinil 0.5DS (5)	6.5	b	17.3	b	28.0	c	58.3	c	54.4	a	97.0	a	33.9	b
Fludioxinil + mancozeb 1DS (5)	5.6	b	20.7	b	57.0	a	98.3	a	49.4	a	93.3	a	31.3	b
Untreated/non-inoculated	50.7	a	100	a	57.2	a	100	a	53.8	a	100	a	12.1	a
Untreated	7.1	b	23.5	b	32.2	c	60.0	c	52.9	a	95.5	a	33.2	b

Potato seed-pieces were either (A) injected with a zoospore suspension of *P infestans* then treated with seed-piece fungicides 48 h after inoculation (replicated twice); (B) cut and inoculated by placing the cut surface onto a mycelial homogenate of rye agar media and *P infestans* (replicated once) (C) potato plants were inoculated with an aerosol spray of a zoospore suspension of *P infestans* (replicated twice), 1997–1998.

[a–d] As for Table 2.

applied when sprouts had broken dormancy. Sprouts averaged 15 leaf initials at the time of application (15.1 ($\pm$0.45), n = 20), with about eight internodes between the base of the sprout and the first emerged leaf. The amount of fungicide required to treat the seed tubers was calculated from manufacturer-recommended seed application rates. Some of the proposed treatments were not pre-formulated for use as potato seed-piece fungicides. Formulations of fungicides intended for use as foliar-applied liquid sprays were used. Rates of foliar fungicides for seed-piece application were calculated from known effective foliar-application rates[11–13] for the control of potato late blight in potato foliage. Fungicides were applied as liquid to the seed-pieces with a carbon dioxide powered boom at a pressure of 550 kPa with one XRI 11003VS nozzle per 1 m wide spray application table at 1 m s^{-1}. The treated tubers were dried before planting. Negative and positive controls were included for comparison. Transmission of late blight from (a) infected seed and (b) seed infected at cutting included inoculated and untreated seed tubers (negative control) and non-inoculated and untreated seed tubers (positive control). The foliar infection experiments included potato plants that developed from seed tubers that were untreated and non-inoculated (positive control).

2.3 Field experiments

The field experiments were planted at the Michigan State University Muck Soils Research Station, Bath, MI on 1 June 1997 and on 25 May 1998, in five-row by five-plant plots, 86 cm row spacing, total 25 plants, replicated four times in a randomized complete block design. Plots were irrigated as needed with sprinklers and were hilled immediately after emergence. No foliar fungicides were applied to either of the field experiments. Weeds were controlled by hilling and with metolachlor at 0.77 kg ha^{-1} 10 dap (days after planting), bentazon, at 0.17 kg ha^{-1}, 20 and 40 dap and sethoxydim at 0.06 kg ha^{-1} 58–60 dap. Insects were controlled with imidacloprid at 1.4 kg ha^{-1} at planting, carbaryl at 1.4 kg ha^{-1}, 31 and 55 dap, endosulfan at 0.98 kg ha^{-1}, 65 and 87 dap and permethrin at 0.56 kg ha^{-1}, 48 dap. The dates of application were similar for 1997 and 1998.

2.4 Data collection

Plant emergence was rated in all experiments. Rate of emergence was calculated as the relative area under the plant emergence progress curve (RAUEPC) with a modification of the method used to calculate the relative area under the disease progress curve (RAUDPC).[14] The AUEPC was calculated by adding the area under the linear progression of number of plants emerged between each successive estimation of emergence from planting to full emergence. The RAUEPC is calculated by dividing the measured AUEPC by the maximum AUEPC (100 $\times$ duration of the emergence period, from planting to full or final plant number emerged).

Plots (field) and individual plants in each treatment (controlled environments) were rated visually for percentage leaf area with symptoms of late blight. The average amount of disease that developed over the disease progress period was expressed as the relative area under the disease progress curve (RAUDPC)[14] which is used extensively to measure disease progress over a given time period.[11–13,15] The area under the disease progress curve (AUDPC) was calculated by adding the area under the linear progression of disease between each successive estimation of disease from inoculation to 100% plant death in the untreated check.[14] The RAUDPC was calculated by dividing the measured AUDPC by the maximum AUDPC (100 $\times$ duration of the epidemic, from inoculation to 100% plant death).

In the controlled-environment foliar-inoculation experiment, the ratio of infected to non-infected leaves was calculated with respect to the position of the leaf on the main stem at 14 days after inoculation. Number of leaves with symptoms was evaluated at each main stem leaf position above the soil surface, and the ratio of infected to non-infected leaves was calculated for each treatment. The first leaf above the soil was counted as leaf one. A leaf was considered infected if it had one or more late blight lesions on any of the leaflets or the petiole. The stem that was apically dominant [(i) had most main stem leaves, (ii) and had the greatest stem diameter] was selected for the evaluation on each of 10 separate plants.

Data were analyzed by two-way analysis of variance and means compared at P = 0.05 level of significance by a multiple range comparison of means (Tukey, SigmaStat). Data were combined in the controlled environment experiments (1997) because the variations between separate treatments in different runs of the experiment remained constant.

3 RESULTS

3.1 Controlled environment experiments

3.1.1 Seed tuber infection simulation and inoculation

Plant emergence did not exceed 40%, except in the non-inoculated untreated control, regardless of the seed treatment (Table 2A). With some of the treatments, fewer plants emerged than the untreated control but differences were not statistically significant (P = 0.05). The rate of emergence (RAUEPC) and number of plants emerged after tubers that were inoculated by injection and treated with any seed treatment was significantly lower than the untreated and non-inoculated control (Table 2A).

3.1.2 Transmission of infection at seed-cutting simulation

The rate of emergence (RAUEPC) and number of plants emerged was not significantly different from the untreated and non-inoculated control or the untreated and inoculated control for any inoculated seed treatment combinations (Table 2B). Of the plants that emerged, some developed late blight lesions on

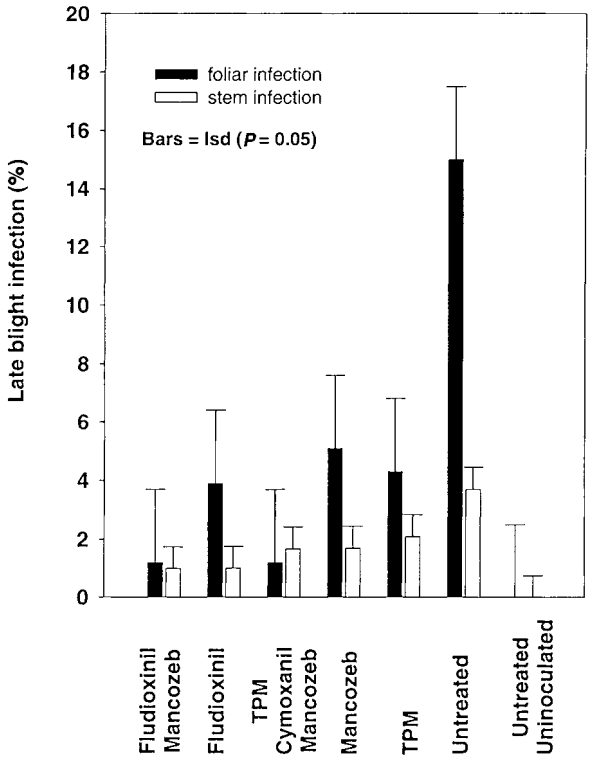

Figure 1. Foliar and stem late blight infection of potato plants that emerged from fungicide-treated seed pieces. Plants inoculated with *Phytophthora infestans* 10 days after emergence and evaluated at 14 days after inoculation (averages of three experiments). TPM = thiophanate-methyl.

the foliage, eg plants that emerged from tubers that were untreated and were treated with fludioxinil, and with thiophanate-methyl + mancozeb, but there were no significant differences ($P = 0.05$) between any treatments (Table 2B). The number of infected plants was generally low. Of the three plants that emerged from 10 planted seed pieces, one had late blight.

3.1.3 Foliar infection simulation and inoculation

Tubers that were not inoculated but were treated with a seed treatment emerged at a rate (RAUEPC) not significantly different from the non-inoculated and untreated control (Table 2C). The number of plants emerged was not significantly different between treatments (Table 2C).

Initial foliage symptoms of late blight were significantly ($P = 0.05$) reduced by all seed-piece treatments 10 days after inoculation in comparison with the untreated and inoculated check; however stem infection was not significantly reduced (Fig 1). The rate of late blight development (RAUDPC) after inoculation over a 21-day period was significantly higher in all treatments in comparison with the untreated and non-inoculated control but not significantly different from the untreated and inoculated control (Table 2C).

The number of infected leaves was determined at each main stem leaf position above the soil surface, and the ratio of infected to non-infected leaves was calculated for each treatment 14 days after inoculation (Fig 2). In some of the plants that developed from treated seed, the lower leaves on the main stem had a lower ratio of infected leaves in positions 1 to about 6 (leaf 1 being the leaf closest to the soil and 6 a mid-stem leaf). The ratio of infected leaves among treatments was similar to the untreated plots above leaf position 6.

Fludioxinil and both treatments containing thiophanate-methyl also had low ratios of infected leaves from the lower canopy (leaves 1–4), (Fig 2). For all treatments, the ratio of diseased to non-diseased leaves was lower in leaf positions above 10 (Fig 2).

3.2 Field experiments

3.2.1 Tuber inoculation experiments

The number of emerged plants that developed from inoculated seed-pieces did not exceed 25%, regardless of the seed treatment (Table 3A). All treatments and the untreated inoculated control had significantly fewer plants emerge in comparison with the untreated and non-inoculated control (Table 3A). No late blight was observed on plants that emerged.

3.2.2 Transmission of infection at seed-cutting simulation

Rate of emergence (RAUEPC) and percentage emergence of plants after the cut surfaces of seed tubers were inoculated and treated with cymoxanil + thiophanate-methyl + mancozeb, and thiophanate-methyl + mancozeb were not significantly different from those of the untreated and non-inoculated control (Table 3B), but were higher than those of untreated and inoculated tubers, thiophanate-methyl- and fludioxinil-treated seed tubers. Fludioxinil + mancozeb, cymoxanil + thiophanate-methyl + mancozeb, and thiophanate-methyl + mancozeb and the untreated and non-inoculated control had close to 100% emergence and had significantly more plants than that of the fludioxinil and the untreated and inoculated control (Table 3B). No plants with lesions caused by late blight were observed after emergence of plants that developed from tubers exposed to the cut face inoculation.

3.2.3 Foliar inoculation experiments

No significant difference ($P = 0.05$) was observed in rate of emergence or final plant stand between any treatments emerged from treated seed tubers 21 days after planting (Table 3C).

Foliar late blight progress (RAUDPC) after inoculation of the immature canopy in plants that developed from untreated and treated seed-pieces (all fungicide treatments) was significantly higher ($P = 0.05$) than with the untreated/non-inoculated control (Table 3C). The plants that developed from treated seed were infected with late blight at a rate that was not significantly different from plants that developed from untreated seed-pieces (Table 3C).

4 DISCUSSION

Potato seed piece treatments are useful for the control of seed-borne diseases such as black scurf and stem

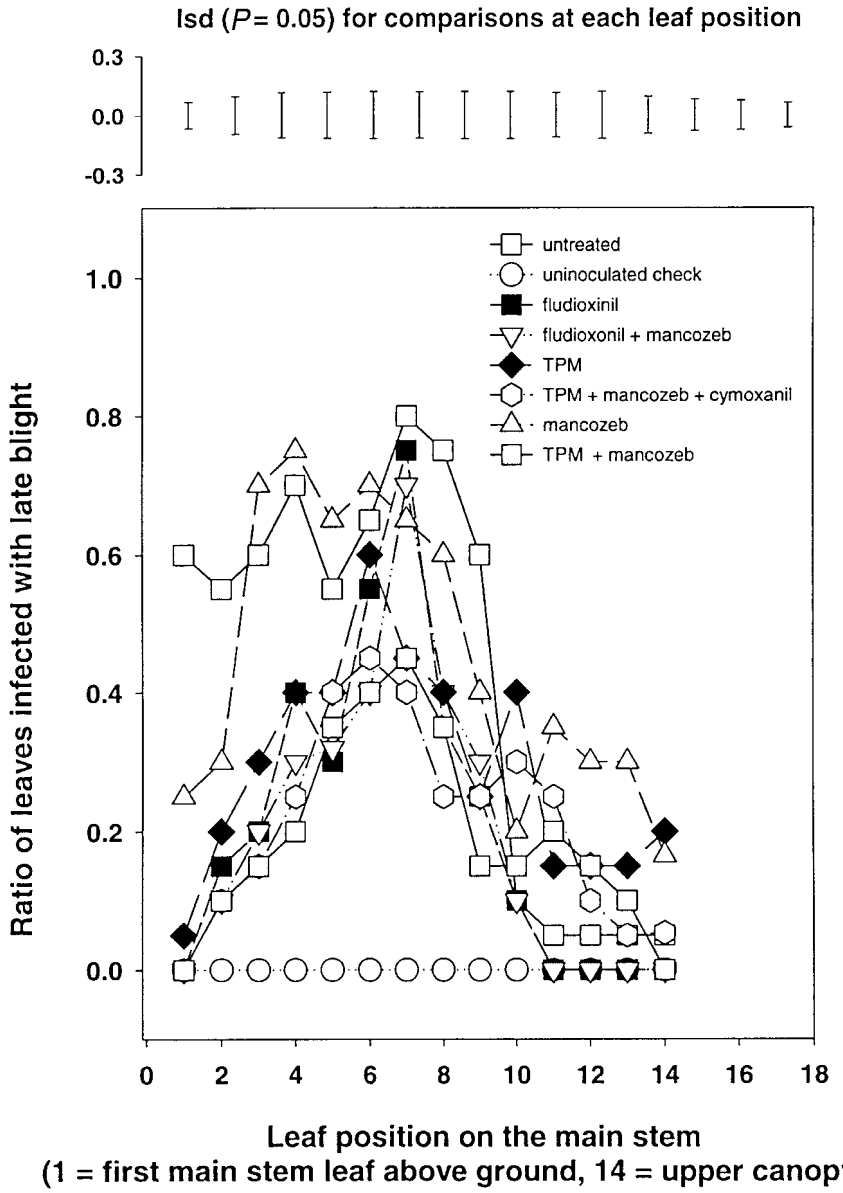

Figure 2. Ratio of infected: healthy potato leaves inoculated with *Phytophthora infestans* in relation to leaf position on the main stem (averages of three experiments).

canker (*Rhizoctonia solani* Kuhn), silver scurf (*Helminthosporium solani* Durieu & Mont) and dry rot (*Fusarium* spp).[5,16,17] These results confirm that the addition of active ingredients that are effective in the management of foliar late blight to existing seed treatments expands their spectrum of activity. The simulation of different phases of infection of potato late blight identified effective treatments for seed-borne late blight and aerial infection of the immature canopy.

Pathogens such as silver scurf, black scurf and dry rot typically rely on seed infection to establish a moderate level of sprout infection, thereby initiating the seasonal epidemic on stems either above or below ground.[18] Severe infection of the sprout with *P infestans* results in premature sprout death and non-emergence.[3] No seed treatments effectively controlled late blight spread inside the tuber. The application of seed treatments to infected seed did not enhance emergence and did not prevent disease developing on some of the emerged sprouts. Internal tuber infections were not controlled with the seed treatments tested. The low number of emerged plants and decreased rate of emergence indicated that sprouts were becoming infected and killed prior to emerging from the soil. The mechanism of infection of sprouts by *P infestans* has not been fully described but a study by Kirk *et al* (unpublished) has shown that infection of developing sprouts on potato tubers is initiated in connecting tissue at the point of attachment of the sprout to the mother tuber.

The development of products for late blight control is currently focused on preventing the transmission of the disease at seed cutting.[7] Simulation of cut-surface spread of late blight from tuber to tuber in this study confirmed that cut surfaces exposed for as little as 30 s

to late blight inoculum are readily infected. The cut surface is readily infected by *P infestans*[7] but is also accessible to the active ingredients contained in the seed treatments. Infection may have been partially inhibited by the mancozeb component of the seed treatments. Mancozeb does not penetrate foliar tissue and the pathogen evades the fungicide once penetration has taken place;[19] mancozeb may therefore not move far from the site of application on tuber tissue. The cut surface of the tuber may allow mancozeb to move sufficiently far inside the cut tuber to prevent the establishment of late blight and prevent the infection of sprouts. The cymoxanil component of, for example, the thiophanate-methyl + cymoxanil + mancozeb seed treatment is systemic in plant tissue[19] and may prevent late blight infection of sprouts in cut seed where the application of the seed treatment is delayed in blight infected seedlots. However, transmission of late blight to cut tuber surfaces in the controlled environment experiments produced plants that were infected with *P infestans*. No treatment gave significant control of this infection, which indicated that the fungicides tested were not fully effective against late blight. The risk of plants emerging from the soil with late blight remains even when seed treatments with components effective against late blight are included in the formulation. The reduced rate of emergence of plants after seed treatments that did not contain mancozeb was evident. A slower rate of emergence may enable the late blight infection to become established and either kill the developing sprout or allow the infection to be carried above ground and initiate a new crop infection. These results confirm other findings[7] that pre-cutting and use of seed treatments (specific and unspecific for late blight) may increase the risk of blight spreading from the seed to the sprout and ultimately to foliage.

Secondary spread of late blight between plants may occur at an early stage in canopy development. The effect of seed treatments may endure sufficiently to help reduce the vulnerability of immature leaves to infection by *P infestans*. Seed-piece treatments reduced overall disease in the immature canopy after foliar inoculation with *P infestans* in controlled environment experiments. This was especially true for leaves near the base of the stem, but not all leaves were protected from late blight. Leaves that had a reduced ratio of infection were at the base of the main stem and may have been exposed to the seed treatments at the time of application. Immature leaf primordia were less than 0.5 cm in length at the time of treatment[20] and fungicides applied to seed may accumulate on tuber structures such as developing sprouts. The accumulated fungicides are likely to be stored, dispersed and taken up (if systemic) by the developing leaf, stolon and root primordia present on these sprouts. Redistribution of active ingredients may result in effective dose accumulations on the developing leaves and internodes. Leaves at the base of the stem in some varieties are less susceptible to late blight infection than leaves close to the flower.[21]

Corresponding field studies designed to examine the effect of seed treatments on subsequent foliar infection did not confirm the controlled environment studies. The quantity of *P infestans* inoculum and conducive environment in the field studies allowed infection of all unprotected foliage and resulted in rapid and complete plant death.

Seed health and quality is of paramount importance to seed and commercial growers in potato crop production.[6] Although it is not possible at present to be completely certain that a potato seed lot is 100% free of late blight, it is prudent to plant seed that meets established seed certification standards. The application of appropriate seed treatments to healthy seed may seem counter-intuitive. However, the potential for late blight to establish itself in young post-emergence foliage before prophylactic fungicide applications begin suggests that additional early-season protection may be warranted in potato-growing areas with a history of late blight. Seed treatment applied to healthy seed may provide some protection against early-season late blight and may persist sufficiently to overlap with the beginning of conventional foliar fungicide applications, providing more complete protection.

Some seed treatment fungicides, such as the thiophanate-methyl + mancozeb + cymoxanil treatments, have systemic and non-systemic fungicide components. These formulations suppressed late blight development in newly emerged plants in controlled environments. The precise timing of application of seed treatments in relation to seed cutting, sprout development and time of planting requires further study.

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